MAYO CLINIC

76-Year-Old Man With Abdominal Pain, Fever, and Maculopapular Rash

Adam C. Bledsoe, MD; Liam Zakko, MD; and Conor G. Loftus, MD

A 76-year-old man with a history of vasovagal syncope and diverticulitis presented to his local urgent care center with abdominal pain, fever, and maculopapular rash of 1 day's duration after receiving the influenza vaccine. Before this illness, he was in his usual state of health. Initially, an acute viral illness was believed to be the cause. His medical team recommended symptomatic treatment.

Four days later, he returned to his primary care physician because of fever as high as 38°C, tachycardia, nausea, decreased appetite, and abdominal pain. History taken at that time revealed travel to Central America 2 weeks before his presentation. Laboratory evaluation revealed the following (reference ranges provided parenthetically): white blood cell count (WBC), 29.5 × 10^9/L (3.5-10.5 × 10^9/L) with elevated neutrophil and eosinophil counts; alkaline phosphatase, 262 U/L (45-115 U/L); aspartate aminotransferase, 35 U/L (8-48 U/L); alanine aminotransferase, 132 U/L (7-55 U/L); bilirubin, 6.9 mg/dL (≤1.2 mg/dL) (predominantly direct); international normalized ratio (INR), 1.7 (0.8-1.2); ammonia, 68 μmol/L (≤30 μmol/L); and C-reactive protein, 50.4 mg/L (≤8.0 mg/L). The patient was hospitalized, and ampicillin-sulbactam was initiated. Results of abdominal ultrasonography were inconclusive for biliary obstruction. Subsequent magnetic resonance cholangiopancreatography revealed no evidence of extrahepatic biliary obstruction.

The patient continued to have abdominal pain, nausea, and jaundice of unknown etiology. Given concern for a drug reaction, his antibiotics were changed to ciprofloxacin, metronidazole, and doxycycline. With his recent travel history, serologic evaluation for *Ehrlichia*, viral hepatitis, cytomegalovirus, *Babesia*, Lyme disease, and malaria was performed but returned negative results.

1. Which *one* of the following is the *most likely* cause of this patient's jaundice?

a. Chagas disease
b. Ampicillin-sulbactam
c. Hemolysis
d. Infiltrative disease
e. Extrahepatic biliary obstruction

Test results were negative for common Central American infections that cause cholestatic liver injury, and involvement of the biliary tree is rare in Chagas disease, especially in the acute phase.[1] Symptoms had begun before antibiotic therapy, excluding ampicillin-sulbactam as the likely cause. Hemolysis can cause jaundice, but elevation is usually seen in the indirect (unconjugated) bilirubin, and this patient had direct (conjugated) bilirubin elevation. Since Chagas disease does not typically cause cholestasis, antibiotics were initiated after the jaundice, the bilirubin is conjugated, and MRCP was negative, infiltrative disease would be the most likely cause of this patient's symptoms in this case. Finally, ultrasonography and magnetic resonance cholangiopancreatography ruled out extrahepatic biliary ductal obstruction.

Infiltrative diseases to consider as an etiology for intrahepatic cholestasis are amyloidosis, sarcoidosis, lymphoma, primary malignant tumor of the liver, or metastatic disease.[2] In this case, without a clear lesion seen on magnetic resonance imaging or ultrasonography, a diffuse process is more likely. With no evidence of biliary obstruction, other causes of elevated liver test results could include primary biliary cirrhosis, systemic inflammation, or drug effect.[2] When a patient presents with elevated liver test results, no clear etiology, and evidence of severe hepatic synthetic dysfunction (elevated INR [>1.5]), the practitioner must consider the possibility of acute liver failure. Such patients should be referred to a liver transplant center.

See end of article for correct answers to questions.

Resident in Internal Medicine, Mayo School of Graduate Medical Education, Rochester, MN (A.C.B.); Fellow in Gastroenterology and Hepatology, Mayo School of Graduate Medical Education, Rochester, MN (L.Z.); Advisor to resident and fellow and Consultant in Gastroenterology and Hepatology, Mayo Clinic, Rochester, MN (C.G.L.).

At this point, the patient was transferred to Mayo Clinic for further evaluation. On arrival, he was oriented to person, place, and time but very somnolent. His temperature was 36.9°C, heart rate was 108 beats/min, blood pressure was 138/75 mm Hg, and respiratory rate was 24 breaths/min. Physical examination revealed a maculopapular rash on his face, neck, back, and abdomen. Laboratory tests revealed the following: hemoglobin, 12.9 g/dL (13.5-17.5 g/dL); WBC, 37.9 × 10^9/L (11% eosinophils); platelet count, 88 × 10^9/L (150-450 × 10^9/L); INR, 1.9; alkaline phosphatase, 149 U/L; aspartate aminotransferase, 29 U/L; alanine aminotransferase, 70 U/L; total bilirubin, 11.6 mg/dL; direct bilirubin, 9.7 mg/dL (0.0-0.3 mg/dL); ferritin, 1217 μg/L (24-336 μg/L); and lactate dehydrogenase (LDH), 517 U/L (122-222 U/L). Serum protein electrophoresis was negative for monoclonal protein, and the tryptase level was normal. Computed tomographic (CT) angiography of the chest, performed to exclude pulmonary embolism, revealed lymphadenopathy of the chest and upper abdomen and splenomegaly. No pulmonary embolism was identified. Transthoracic echocardiography revealed a left ventricular ejection fraction of 47% with posterior and apical regional wall motion abnormalities.

2. Eosinophilia can be associated with many disease processes, but it would be *unexpected* in which *one* of the following diagnoses?

a. T-cell lymphoma
b. DRESS (drug reaction with eosinophilia and systemic symptoms) syndrome
c. Eosinophilic granulomatosis with polyangiitis
d. Parasitic infection
e. Adult Still disease

There are many causes of secondary hypereosinophilia, including malignant neoplasms, drug reactions like DRESS syndrome, parasitic infections, and autoimmune disorders like eosinophilic granulomatosis with polyangiitis.[3] These processes must be excluded before evaluating a person for a primary hypereosinophilic syndrome or a clonal eosinophilia.[4] Adult Still disease can present with symptoms similar to this patient's, but eosinophilia is not a typical finding in adult Still disease,[3] making it the least likely cause.

Given the complexity of this case, additional consultations were requested. Colleagues from the infectious disease service recommended extensive work-up, which yielded negative findings. The dermatology service was consulted and performed a skin biopsy of the maculopapular rash. Pathologic examination of the specimen revealed neutrophilic dermatosis. Given the rash, hypereosinophilia, and negative results on infectious disease evaluation, prednisone was initiated and titrated to a dose of 100 mg/d. The patient's cholestasis and eosinophilia initially improved with the prednisone.

3. Which *one* of the following is the *most likely* cause of this patient's neutrophilic dermatosis?

a. Pyoderma gangrenosum
b. SAPHO (synovitis, acne, pustulosis, hyperostosis, and osteitis) syndrome
c. Sweet syndrome
d. Behçet disease (Silk Road disease)
e. Rheumatoid neutrophilic dermatosis

All of the listed diagnoses have biopsy findings consistent with a neutrophilic dermatosis.[5-7] The clinical scenario helps narrow the differential diagnosis. Pyoderma gangrenosum is a neutrophilic dermatosis with slowly expanding ulcerated plaques.[5] Our patient did not have these physical findings. SAPHO syndrome is a disease with components of synovitis, acne, pustulosis, hyperostosis, and osteitis.[6] Neutrophilic pseudoabscesses are common with SAPHO syndrome,[6] but this patient had no other features of this syndrome. Sweet syndrome is the most likely cause of this patient's neutrophilic dermatosis because it involves an acute widespread rash and fever. It is associated with an underlying systemic disease in 35% of cases, often a hematologic malignancy.[5] Patients with Behçet disease (Silk Road disease) can have a widespread neutrophilic rash, but it is usually accompanied by oral, ocular, and/or genital lesions.[7] Rheumatoid neutrophilic dermatosis is seen in patients with advanced rheumatoid arthritis.[5]

Given the association between Sweet syndrome and hematologic malignancy, hematologic evaluation was performed. Positron emission tomography–CT revealed diffuse fludeoxyglucose F 18 uptake in the patient's lymph nodes, spleen, and bone marrow.

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